ECLAMPSIA and its TREATMENT
from the standpoint of the General Practitioner
or principally from the Clinical Aspect

by

R. MACDONALD ROBERTSON, M.B., C.M.

------oOo-----

ECLAMPSIA and its Treatment viewed from the standpoint of the General Practitioner or from the Clinical Aspect rather than from that of the Morbid Anatomy.

INTRODUCTION.

Ever since the opening of a new era in the 17th century by the introduction of Midwifery Forceps, the risks and dangers naturally attendant on pregnancy, labour and the puerperium have been gradually diminishing. As a result of prolonged investigation and various discoveries from time to time of illustrious accoucheurs and other men of science, such risks and dangers as the so-called "child-bed fever", ante and post partum haemorrhage, unusual and abnormal presentations, a too narrow outlet at the brim of the pelvis, threatened rupture of the uterus and other risks are much more successfully treated at the present day than even forty or fifty years ago, chloroform and antiseptic surgery in great measure contributing to those most desirable achievements. As a consequence the present mortality compared to what it was formerly must have diminished very considerably.

We are still, however, confronted by a very formidable and somewhat mysterious danger, viz:- Eclampsia, the subject of this thesis. Happily in our time signs are not wanting that it too, like the former risks and dangers to which I have just alluded, may soon be relegated to the limbo of conquered

diseases of the past.

This hopeful outlook is based upon the circumstance that during recent years our knowledge of the physiological and pathological processes of the pregnant state is being much widened, so that now, more especially from a prophylactic point of view, we are much better able to anticipate and counteract its dire occurrence.

FREQUENCY.

Opinions vary regarding the frequency of its occurrence. Most statistics have been taken from Hospital Reports. Certain authorities state the proportion as 1 in 133 cases, others as 1 in 330, and others as 1 in 500. Again, it would appear that "Eclampsia varies markedly in frequency at different times". According to Tarnier's clinic in Paris in 1872, one case occurred in 47 labours, in 1882 one in every 730, and in 1891 one in every 130 - a somewhat remarkable variation.

In the Edinburgh Royal Maternity Hospital, according to Dr. R. W. Johnstone in an article in the Journal of Obstetrics and Gynaecology for January 1910 ~~1910~~, in 5,800 cases he found 126 of Eclampsia. In the Rotunda Hospital, according to Dr. Jillett, one case occurred in 332.16. Hospital reports, however, probably give an exaggerated percentage of Eclampsia cases as compared with labour cases universally, as doubtless it is the custom to transfer many threatened cases in private practice to the nearest Maternity Hospital when there is a prospect of this being accomplished safely and in time, in order to allow the patient to have the benefit of the constant supervision and observation obtainable in such institutions which the busy general practitioner can ill afford to bestow on account of the limited time and

resources at his disposal. It is more important to ascertain the proportions of cases occurring before, during, and after labour. I have copied the following statistics of this latter question from Dr. Whitridge Williams' Obstetrics:-

	Antepartum	Intrapartum	Postpartum
Olshausen	40%	46%	14%
Knapp	24.5%	60.9%	14.6%
Goldberg	26%	57%	17%
Green	36%	22%	42%

According to these statistics, with the exception of those recorded by Green, the greater proportion of cases occurs during labour and the least during the puerperium. This view is supported by statistics from other sources. From 316 cases recorded by Dr. Haultain, 62 occurred antepartum, 190 intrapartum and 64 postpartum. Of the 126 cases referred to by Dr. R. W. Johnstone, 16 occurred postpartum or 12.6%.

From my own experience in a number of about, speaking roughly, 2,200 confinements, I have had 7 well marked cases of Eclampsia; of these, in 3 cases Eclampsia commenced before labour, in 3 cases during labour, and in 1 case after labour.

I would especially draw attention to the circumstances of the greater proportion of cases occurring during labour, the significance of which will be

See page 43

considered later on; also to the fact that a certain proportion of cases does occur during the puerperium. As we shall see this has an important bearing on the placental cell theory of Eclampsia. Page 19 & 34

Authoritative statistics give about 80% of Eclamptic cases as primipara and about 20% as multipara. Of my 7 cases, 4 were primipara and 3 multipara; very young primipara on the one hand and aged ones on the other are held to be more liable. One of my primipara was 36 years old, the others ranged from 24 - 27 years old. One of my multipara was 26½ years old and had had one previous confinement safely; another was 24 years old and had had one previous confinement, also safely. The other was 42 years of age and had 9 previous confinements without serious complications. Cases of multiple pregnancy are also considered to be more liable; all my cases were single births. I may here state that probably I would have had a larger number of Eclamptic cases had not prophylactic measures been adopted in certain albuminuric cases occurring near the termination of labour, a circumstance which I consider affords a favourable omen as to the future outlook.

NATURE OF THE MALADY.

Older Views. Regarding the nature of this so-called "Eclampsia" (from the Greek "eklampein" to shine forth), several theories during the past "have had their day and cease to be". Of these it will suffice to refer briefly to only a few.

(1) Morbid cerebral conditions occurring towards the termination of pregnancy were considered by some as the cause of Eclampsia. Such were cerebral anaemia, cerebral congestion, and cerebral oedema. None of such conditions, however, will account for Eclampsia.

(2) Exalted Nervous Tension was the idea propounded by Dr. Barnes. He suggested that extra nervous energy might be gradually stored in the system during pregnancy to be utilised for the purpose of stimulating and facilitating labour when the time for that had arrived, but if by then the blood was to a certain degree poisoned by insufficient elimination, the result was an explosion of this stored up energy resulting in the convulsions of Eclampsia. Of course such a vague theory must rest entirely on supposition, as we have no proof of storage of nervous energy, and if we had, such is incapable of measurement. Besides, clinical observation would appear to support a directly opposite view.

(3) A Bacterial Origin of Eclampsia has been held. Hergott actually believed Eclampsia to be

contagious. This notion probably arose from those occasional instances where the occurrence of the malady assumes an apparently epidemic nature and a series of cases takes place in the same locality about the same time. No bacterium capable of producing the disease has, however, been isolated. Moreover, this theory would hardly explain the greater liability of primipara to be attacked.

(4) Renal inadequacy alone was considered to be the true cause arising from a primary blocking and consequent defective function of the kidneys occurring during the later months of pregnancy with a tendency to a gradual increase in severity whereby the organ was progressively rendered more and more inadequate to eliminate certain used up and waste products of metabolism, especially urea. Those products retained in the system accumulated and acted as toxins especially affecting the central nervous system, ultimately giving rise to the dreaded symptoms characteristic of Eclampsia. Among the various theories for the causation of this renal perversion, probably the chief one was a mechanical blocking of the ureters and renal pelves arising from the pressure of the increased bulk of the abdominal contents due to the enlarged uterus, thus interfering with the passage of urine and its constituents into the bladder. The greater

liability of primipara to suffer was probably considered as the outcome of the greater force necessary to overcome the rigidity of the abdominal walls hitherto unrelaxed, than what would obviously be necessary on subsequent occasions.

Due reflection, however, would render this theory untenable as it came to be observed that similar abdominal enlargements by tumours, such as fibroids or ovarian, were not attended by Eclampsia. Moreover, the death of the foetus in utero or its delivery in not a few cases resulted in either cessation or marked improvement of the symptoms. Later investigation has revealed the fact that other organs of excretion and metabolism are simultaneously, if not primarily, involved and that in all probability the morbid condition of the kidney is the result partly of the excessive work thrown upon it by the failure of these other organs, especially the liver, partly of excessive vaso-constriction of the renal arteries and arterioles obstructing the blood supply to the glomeruli, the result of a toxin or toxins, and partly in extreme cases due to the excess of such toxicity in the blood tending to cause degeneration of the renal tissues by giving rise to autolysis.

Reference has already been made to the former theory of urea being considered the most important poison. At a time when its properties were little

understood, the view was held that urea, when retained in the system, was the special toxin that acted upon the brain cells and gave rise to Eclampsia. Of course we are now aware that not only is it improbable that urea is capable of acting as a toxin at all, but that in Eclampsia its production is either suppressed or affected to a very small extent and that when present in the blood it acts as a powerful diuretic.

(5) Retention of the Urinary Constituents as a Whole acting as a Poison. This idea arose from the observed fact of the fits occurring with the suppression of the renal function and of the return more or less to health on the restoration of this function. Now doubtless, as we shall see, Eclampsia is due to an accumulation of some special toxin or of several such in the system, but it is highly probable that these toxins as such are incapable of elimination by the kidneys, therefore the normal urinary constituents by their very capability of elimination cannot, on the above hypothesis, be the toxins.

(6) Ordinary Acute Nephritis has been held to be the cause of Eclampsia, but microscopic examination reveals the fact that the latter is not a true nephritis but simply an acute degeneration of the renal epithelium especially of the convoluted tubes

characterised by a fatty infiltration. And many patients who really suffer from nephritis pass through the term of pregnancy, labour and the puerperium without suffering from Eclampsia. There is, however, a marked resemblance clinically between Eclampsia and uraemia, and although the primary pathological condition in the two cases is quite different, the ultimate exciting effect on the brain cells may be of the same nature, the final result in both cases being, at least from the clinical aspect, almost identical, viz: the occurrence of convulsions and coma due to some toxin or toxins which in neither case has as yet been identified. A marked difference, however, does exist between Uraemia and Eclampsia, and that is that Eclampsia is attended with Pyrexia, while with Uraemia there is none. The prognosis of Eclampsia is doubtless more grave in patients who have been suffering from nephritis. Should Uraemia and Eclamptic convulsions occur simultaneously, the prognosis would probably be hopeless.

Such were some of the most prominent theories regarding the causation of Eclampsia, theories now found to be untenable in view of the increasing light shed at the present day on the physiology and pathology of the various functions of the body.

Modern Theories. The modern views regarding the nature of this malady will now demand our attention. The foundation of these is based upon the result, as we have stated, of more minute and correct investigation of the physiological and pathological condition of the state of the tissues, especially of the principal organs concerned with metabolism. This investigation has revealed the existence in toxaemia of pregnancy of a tendency to extensive thrombosis and widespread degeneration leading to necrosis more or less of all organs concerned with metabolism, especially of the liver, indicating the invasion of some special toxin or toxins. To quote the words of Dr. Herbert French, "It is more than likely that certain cases of pregnancy give rise to a poison of some kind which in turn is the cause of nephritis". Regarding the origin or nature of this poison, there are more theories than one.

(1) Thyroid inadequacy. Increased knowledge of the thyroid system would seem to indicate that its importance in pregnancy is greater than formerly supposed. It is now known that the thyroid or more especially the parathyroid secretion exercises an important function in regulating the general metabolism of the body. When impairment of these organs occurs, interfering with their secretion, metabolism is retarded; when arrested altogether

as in cases of excision, metabolism ceases. Moreover in those cases where its function is in abeyance, when it is administered artificially, metabolism regains its efficiency and continues so long as this administration lasts. In cases of myxoedema where the thyroid secretion is deficient, a peculiar train of symptoms occurs bearing a certain similarity to those preceding and ending in Eclampsia. There is tendency to lassitude, mental deterioration, extensive oedema of surface of the body of such a solid nature as to resist pitting on pressure; there is more or less anuria, decreased secretion of urea, and occasional attacks of albuminuria, and in certain cases of excision of the thyroid death has been preceded by tetanus and convulsions.

Lange first drew attention to the significance of the thyroid enlargement observable in a large number of cases of pregnancy. He administered in such cases thyroid extract, with the result of diminution of the hypertrophy and in cases of nephritis occurring in pregnancy diuresis was established after the administration of iodothyrin.

According to Nicholson and others, the modus operandi of this effect of the thyroid system on metabolism is probably on account of its vaso-dilatory function being a directly opposite one to

that of the supra-renal gland and thus acting as an opposing factor to the latter, the function of which is that of a vaso-constrictor. It would thus appear that the functions of the thyroid system and the supra-renal gland being antagonistic one to another normally keep the metabolism of the system properly balanced. From this hypothesis Nicholson has advanced the ingenious theory regarding the causation of Eclampsia, that in the lowering of the thyroid function the supra-renal one gains the ascendency and not only tends to cause widespread constriction of the systematic arterioles, thus raising the blood pressure, but in addition spicial constriction of the renal arteries and arterioles, thus restricting the flow of blood to the glomeruli whereby anuria ensues, with the result of accumulation of waste products in the system from which originate the toxins giving rise to Eclampsia.

Whatever the modus operandi by which the thyroid secretion favourably influences metabolism, I think we may thus far assume as established the theory that when it is deficient or impaired in quality the liver fails to convert certain nitrogenous products into urea which in the pregnant state by their presence in the blood are, to say the least, closely connected with the formation of the toxins which produce Eclampsia.

From the above considerations, it appears reasonable to conclude that the increase in size of the thyroid so often met with during pregnancy indicates the strenuous endeavour of that organ to put forth greater ~~force~~ force to deal effectually with the growing demand for increased metabolism.

Proof of this is afforded by the beneficial effect, amounting in certain cases to absolute cure, which often follows the administration of thyroid in the treatment of the so-called pre-eclamptic state and even of Eclampsia itself in certain instances.

Personally I have not as yet had sufficient opportunities for testing the effects of its administration in cases of Eclampsia, but may mention the case of Mrs S — age 37 years, primipara, about 7 months pregnant whose urine I found on testing in January last to contain a small trace of albumin. I first after administering a calomel purge, put her on a strictly milky diet and gave a diuretic of acetate of potash about 25 grains thrice daily for about one week. At the end of that period the urine was found to be in the same slightly albuminous condition. I then put her on thyroid extract 5 grains thrice daily and had the satisfaction of finding at the end of 7 days of this treatment entire disappearance of the albumin from the urine.

She has since then been delivered at full term of a living child and has made a good recovery without with the exception of a slight attack of phlebitis any return of the albuminuria.

I may here be allowed to refer to the very instructive case related in a publication by Dr. Nicholson of a Mrs S -- of Kirkcaldy in 1901 whom Dr. Henderson of that town treated successfully by the administration of thyroid gland extract, as well as one or two other cases recorded in the same publication and similarly treated with a successful result. Such instances strongly support the idea that sufficiency of thyroid extract plays an important part in keeping in check accumulation of toxins which would inevitably result in Eclampsia.

(2) Transmission of foetal products to the maternal circulation. A certain degree of plausibility is attached to this theory from the circumstance of the improvement which often follows the removal or death of the foetus in utero, and apart from the idea of any special poison being generated from the infusion of the foetal blood with the maternal, it is doubtless the case that an excessive strain is thrown upon the maternal organs of metabolism in the endeavour to eliminate the waste products resulting from foetal katabolism. Of late, however, certain obvious reasons have been the means of discarding this theory as one sufficient to

account for Eclampsia in every instance of its occurrence. (First) we do have often a continuance of Eclampsia after the removal or death of the foetus in utero; (second) Eclampsia does often appear de novo during the puerperium; and (third) what is supposed to be the most striking condemnation of the theory is the occurrence sometimes of Eclampsia during the existence of a vesicular mole first pointed out by Hitschman. ~~After all, however, such a condition may not be contradictory to the theory of foetal products being the cause of Eclampsia as would appear at first sight, as in vesicular mole there is an enormous increase of syncytium and portions of this gaining access into the maternal blood may aid the accumulation of waste products and thus encourage the tendency to Eclampsia.~~ These and other considerations have given rise to the idea that the term Eclampsia may include different types of the malady arising from different causes of which transmission of foetal products may be one.

(3) Placental theory, viz: that the passage of placental villi or parts of villi or syncytium into the maternal blood gives rise to Eclampsia either directly or by producing certain chemical or mechanical changes from which the special poison is developed. This subject has given rise to much discussion. Numerous experiments have been tried to

determine the existence or otherwise of a specific toxin capable of producing Eclampsia both in the normal and the eclamptic placenta.

Veit professed to have discovered as the outcome of placental cells in the blood, antibodies which he termed syncytiolysins which, if in sufficient numbers, counteracted the effect of a specific poison introduced by the transmission of the placental cells into the blood. Should there be a deficiency of such syncytiolysins, the toxin introduced by the syncytium prevailed and Eclampsia was the result.

Ascoli held that the syncytiolysins themselves which were left free over and above what had been used up in dealing with the syncytiotoxins were the cause of Eclampsia.

Weichardt appears so far to agree with Ascoli, but held in addition that the syncytiotoxins liberated by the excess of the syncytiolysins, if not too numerous, were counteracted by antitoxins formed in the blood.

It would be out of place here to enter into a description and discussion of the various intricate experiments by which those observers professed to arrive at their theories.

Leipman failed to obtain the same results as Veit and Weichardt, but professed to have discovered another reaction, viz: that of precipitans.

Subsequent investigations based upon more exact experiments, especially by Wormer Pollak and Frank, have failed to confirm the above results.

The latest investigations appear to support the theory that the poison is a nucleo-proteid liberated from the cells which acts by causing the widespread coagulation of the blood known to exist in Eclampsia.

In addition to this tendency to coagulation, there is of course also the widespread degeneration leading to necrosis of the tissues of important organs. To account for this latter condition it has been suggested that the degeneration is a result of the thrombosis, but both appear to occur simultaneously and now autolysis of the placental cells is looked upon primarily as a more probable cause (Holland).

In placental autolysis several ferments take part, but the most active in producing this degeneration is considered a proteolytic one. Products from this are supposed to circulate in the maternal blood and in turn incite the ferments of cells in other important organs to autolysis also, hence the degeneration and necrosis. To render the above autolytic theory clear, it has to be borne in mind that each cell possesses various ferments peculiar to itself. Normally the function of these ferments is to preserve the cellular vitality by transforming

certain extraneous products into nourishment for the cell as well as the elimination of the used up cellular products. In health these ferments do not attack their own cell; it is otherwise, however, when they are overstimulated or when the cell vitality is low, e.g. in cases of widespread thrombosis or when the products of autolysis of other cells present themselves, then the ferments turn round as it were and attack their own cells and thus effect this degeneration. It is held that placental autolytic products may also lend an additional factor in effecting the coagulation of the blood by setting free more nucleo-proteid.

Such appear to be the latest views regarding the primary cause of Eclampsia, that is the cause leading to the thrombosis and degeneration. A difficulty in accepting such theories evidently arises Page 495 from the circumstance of Eclampsia often occurring de novo during the puerperium after expulsion of the placenta. In the article on postpartum Eclampsia by Dr. R. W. Johnstone already referred to, this objection to the placental origin theory has been ably discussed, and, as he points out, the only view capable of fitting in with this theory which would embrace cases of "placental" Eclampsia is that of Ascoli, viz: that when there is an excess of the antibodies or syncytiolysins over and above those necessary for the destruction of the placental element in

the maternal blood or when the latter is absent, then the syncytiolysins act as the poison. One circumstance especially appears to me to weigh against the placental theory and that is the very rare occurrence of Eclampsia in the case of vesicular mole.

If the theory of the presence of syncytium in the maternal blood as the cause of Eclampsia be the correct one, why do we not have it oftener in vesicular mole when there is developed enormous hypertrophy of syncytium? On such a hypothesis one would almost expect Eclampsia to occur in every case of vesicular mole. On the contrary only one or two instances have been recorded. We shall refer to this subject later on.

Granting, however, the correctness of the placental cell theory, including its autolysis, we have only as yet arrived at the primary or initial cause of which the maternal thrombosis and degeneration are the supposed results, we have still to find out how this leads up to the more direct or immediate cause of the eclamptic phenomena and as a result of further investigation we shall be led to recognise a new class of toxin or toxins, and it is in this later investigation that the ~~"Clinical Picture"~~ clinical symptoms will be our chief guide.

The chief outstanding features clinically we find to be increased blood pressure, anuria, albuminuria, and, what is of most importance, the deficiency

of urea in the urine, this last indicating diminished production of this product by the liver, in other words, a profound disturbance of metabolism.

The correct theory regarding the origin of urea in normal health appears at last to have been solved, and that is, its derivation from certain so-called cleavage products termed ~~ammo~~ amino-acids, the ultimate digestive analysis of the protein of the food, and effected principally by the agency of a pancreatic proteolytic ferment. Roughly speaking, by means of various digestive agencies, the original protein is so acted upon that it is converted into the successive stages of (1st) Acid Albumen, (2nd Proteoses, (3rd) Peptone, (4th) ~~Ammo~~ Amino-Acids. The greater part of these ~~ammo~~ amino-acids are conveyed by the portal vein into the liver and there are converted into urea, which is finally eliminated by the kidneys, only a very small and fixed quantity of these ammo-acids being utilised for the repair and structure of the protoplasms of the tissues. It would thus appear that the greater part of these ~~ammo~~ amino-acids apparently serve only to act as a strain on the digestive organs and on the metabolism of the liver. Thus it is of little or no significance how much or within certain limits how little nitrogenous food we ingest, the supply required for the structure and repair of the protoplasm is, as already stated, fixed and constant and

of comparatively small amount. There is no storing up in the system for future use of these nitrogenous products as is the case with the fats and with material derived from the carbohydrates. In passing I may remark that this circumstance is accountable for the opinion of certain dietists that our ordinary food should contain much less butcher meat than is customary. Whether this view be correct or not, it has, as we shall see, a most important bearing on one item of the treatment of Eclampsia.

To return to the subject on hand. In Eclampsia we have either entire failure of production of urea or marked diminution in the amount produced, indicating hepatic incompetence to transform the poisonous Amino ~~ammo~~-acids into it. These untransformed nitrogenous products are probably to a large extent incapable of elimination by the kidney and are consequently as such retained in the blood, although occasionally they are present in the urine. Is therefore the poison which gives rise to Eclampsia to be found among those retained and unconverted Amino ~~ammo~~-acids? Or if not the direct cause, are they not intimately connected with the causation of Eclampsia? A reply in the affirmative would appear to be justified by the following consideration, viz: that in other diseases where failure to reach the stage of urea occurs symptoms similar and often almost identical to those

of the final stage of Eclampsia take place, e.g. in acute yellow atrophy of the liver where the urea production is seriously interfered with death is usually preceded by coma and often convulsions. Again in acute nephritis especially of scarlatina where there is also marked diminution of urea formation, we have coma and convulsions. The fact of the morbid anatomy of these maladies being different does not affect the argument, the notable feature being that in all three instances there is failure of the kidney to eliminate the proper urinary constituents, and there is especially the absence of urea. Thus far the condition of Eclampsia, acute yellow atrophy, and acute nephritis are identical and for the time being produce identical results. The same effect, but not so well marked, viz: diminished urea resulting in coma before death, occurs in phosphorus poisoning.

To recapitulate - certain morbid conditions occur in which analysis of proteins has been arrested at a stage before that of urea has been reached, the products of this intermediate stage, the ~~amme~~ amino-acids, being incapable of renal elimination, are retained. This condition in the various maladies in which it occurs is always followed by markedly similar nervous phenomena, which, on the other hand, are invariably absent when there is adequate elimination of urea. Such a state of matters certainly appears prima facie

strongly confirmative of the contention that the final or direct cause of Eclampsia is, to say the least, closely connected with the presence of the ~~ammo~~ amino-acids in the blood.

Another theory accountable for the final cause of Eclampsia, however, suggests itself from the widespread condition of coagulation of the blood and the increased blood pressure, and that is the probability of mechanical pressure on the brain cells by either small emboli, small patches of haemorrhage, or by the occurrence of small cerebral aneurisms. The presence of small emboli and haemorrhages is confirmed by postmortem examination.

My reason for supposing the probable existence sometimes of small aneurisms, although I am unaware of its confirmation by pathological examination, arises from the circumstance that in one of my cases of puerperal Eclampsia there also occurred an extensive purpuric rash. Fortunately recovery followed, so that I had not an opportunity of verifying the diagnosis of aneurisms; but in a case of acute rheumatism attended by a medical friend there also occurred an extensive purpuric rash. Death in this case was preceded by convulsions and coma and the postmortem examination revealed the occurrence of numerous small aneurisms. Now although this was not a case of Eclampsia, and certain features clinically

were of course different in the two cases, yet as convulsions, coma and rash were characteristic of both, I consider that there may be some ground for concluding that as aneurisms were found in the rheumatic case, they might also have occurred temporarily in the Eclamptic one.

From the above considerations and taking into account the constant association of Eclampsia with the presence in the blood of untransformed ~~ammo~~ amino-acids as well as with the widespread thrombosis and high blood pressure, two theories, as we have seen, present themselves, either separately or in combination,- (a) that the occurrence of the special toxin is to be found among the ~~ammo~~ amino-acids or intimately associated with them, (b) that the toxin is a mechanical rather than a chemical one, causing cerebral pressure due to either embolus, haemorrhages, or aneurisms. So much for the more direct or immediate cause of the toxicity leading to the convulsions.

To revert again to the first or primary cause leading to this more immediate one, I think there is little doubt, so far as our present knowledge carries us, that autolysis of the hepatic tissue is accountable for the failure of the liver to convert the ~~ammo~~ amino-acids into the non-poisonous urea; but whence again arises the cause of this autolysis? We have already considered the theories adduced to account for this of syncytium and of products in the maternal blood derived from placental cell autolysis as well

as nucleo-proteid also of placental origin; but as we have seen, the ascertained facts of the investigation, so far as they have been determined, do not as yet warrant such an assumption except we adopt Ascoli's view which is based chiefly on circumstantial evidence. The sole reason for such theories appears to have arisen from the discovery of certain placental elements in the maternal blood. But why accept theories of doubtful import, even although based upon a certain degree of circumstantial evidence? Are there not good reasons for concluding that, apart from any placental theory, the intensified metabolic activity incidental to the pregnant state may not of itself in certain instances be sufficient to give rise to the autolytic degeneration of the cells as well as to the thrombosis? I venture to submit that good grounds for such a supposition exist and that such appear to be demonstrated by taking a clinical survey of what we might term the life history of a case of pregnancy revealing the gradual inner working or physiological and pathological processes during the whole term of pregnancy. The more light that can be shed on these processes, the more likely are we to arrive at a satisfactory solution of the subject. In taking such a clinical survey, important phenomena come under notice. For our purpose we will briefly consider a few of the more important as far as

possible in their chronological order, and as a result we will find evidence of excessive strain on the metabolic processes of the system gradually increasing as labour advances and ultimately tending to the failure of urea production and consequent accumulation of ~~ammo~~ amino-acids and other nitrogenous waste products, as well as tendency to widespread coagulation of the blood, and that this condition is not due primarily to any poison of placental origin, but to certain changes which occur in the maternal organism itself.

These important phenomena which will now engage our attention are as follows:- (1) The formation, growth, and gradual development of the foetus and placenta, events more or less underlying the whole gestation period.

(2) The suppression of the menses.

(3) Gastric disturbances and other pathological phenomena.

(4) Secretion of milk.

(5) Increase of cardiac area and other circulatory phenomena.

(6) Enlargement of the thyroid system.

(1) The existence and gradual development of the foetus cannot be maintained without constant repair of its cells and constant elimination of debris. This is impossible without the aid of the maternal

organism, involving increased exertion and strain of metabolism in casting off the foetal waste products in addition to its own. It is quite conceivable that in certain instances, especially in cases of plurality of foetuses, where there is excess of such waste products, that the metabolic strain may prove too much, involving non-elimination of such products and by their accumulation causing autolysis of these organs involved. Such a state of matters will be evidently more liable at a comparatively advanced stage of the growth of the foetus.

(2) I consider that it is also self-evident that suppression of the menses must contribute an additional factor to the maternal strain of metabolism. Normal menstruation in the non-pregnant in addition to its function of casting off part or whole of the then useless decidua mucosa, must also clear the system of waste products which otherwise would have acted deleteriously. I think that proof of this is afforded by the not unusual occurrence of baleful effects occasionally following suppression of the menses from other causes than pregnancy. We have such symptoms as lassitude, severe headache, mental lethargy, dyspepsia, and in some cases, more especially about the age of puberty, we have in addition instances of thyroid hypertrophy and even epileptic attacks, in fact, symptoms not unlike those observed

in toxaemia of pregnancy and Eclampsia.

Of course the cessation of the menstrual flow and the retention of its sanguinary contents during pregnancy probably fulfil at least a two-fold function, (1) the retention of the decidua for the reception of the impregnated ovum as well as a bed for its future development, (2) the retention of the nourishment in the increased supply of blood to meet the extra and growing demands of the occasion, e.g. the growth of the foetus, the enlarged maternal organs of metabolism, the secretion of milk, etc. This, however, entails at the same time the unavoidable retention of waste products in that blood and in consequence an additional extra strain on the eliminating organs in dealing with such products, thus favouring in severe cases their retention in the system and the consequent liability to autolysis.

(3) Digestive disturbances and other pathological phenomena. In the vast majority of cases of pregnancy, such disturbances occur and are exhibited in varying degrees of intensity, ~~and~~ the most prominent and constant being sickness, especially in the earlier part of the day, often accompanied by salivation and constipation. When of a minor degree such symptoms are hardly considered pathological; doubtless, however, they indicate a degree below

the normal standard of health, and as such are the first outward manifestation of a commencing disturbance of metabolism bearing evidence that the salivary glands, stomach, liver and pancreas are fulfilling their function with a certain degree of difficulty. From this we are led to conclude that the blood furnishing these organs with their digestive secretions is beginning to be laden with toxins. In the stomach there is probably excess of hydrochloric acid and defective supply of pepsine or pepsine weak in quality. Vomiting of this excessive acid with partly digested or undigested food is considered by some as a vicarious elimination. Or rather, in order to aid the overstrained metabolism, Nature appears to interfere by inducing a feeling of nausea restraining the appetite and thus diminishing for the time being the quantity of food introduced into the system whereby the accumulation of nitrogenous products in the blood is diminished and metabolism tends to be relieved of the excessive strain.

The foundation of these early digestive disturbances is evidently due chiefly to the commencing failure of the liver to convert into urea the amino-acids, this in turn being due to the tendency even now to autolysis of the hepatic cells by the unusual accumulation of both maternal and foetal waste products; the blood being thus intoxicated

supplies the defective digestive secretions already alluded to, to the alimentary tract. Such a condition occurring at this early stage must be at a time long before placental autolytic products can have accumulated in the blood to such an extent as to have any influence, however possible it may be that such an influence is exerted later on.

Should such a condition remain unrelieved, accumulation of such toxins will increase, digestive disorders will become worse and the condition known as "pernicious vomiting" of pregnancy may ensue. Should the patient survive so far such a serious condition, it is probable that Eclampsia of a highly intractable nature, ending in death, will be the result.

Thus, without having to fall back on the theory of placental autolytic products circulating in the maternal blood, the ever increasing accumulation of waste products appear to me to be of themselves ample to account for the widespread thrombosis, as well as the postmortem appearances of the liver and other organs consisting of haemorrhages and necrosis and even for the occasional occurrence of acute yellow atrophy - hence the non-production of urea and accumulation of ammo-acids.

Fortunately such a disastrous train of symptoms forms the exception to the rule. We have, however, more occasionally, cases of less severity but

nevertheless sufficiently so, as to tax to the utmost the resources of the general practitioner. Happily in the majority of cases, the liver, and following in its train the other metabolic organs, is enabled to rise to the occasion. The accumulation of toxins is checked by their transformation into urea, diuresis is thereby established and the waste products which go to form the urinary constituents are eliminated. This fortunate termination may result from more causes than one. It may be that latent powers hitherto undeveloped in the liver now assert themselves, or that through the benign influence of increased thyroid or parathyroid function it may take on a new lease of life. On the other hand, new resources may arise at the opportune moment, such as even the vomiting itself, a circumstance already referred to, or it may be a substitution of a form of diet less liable to the formation of toxins, e.g. milk, a resource now universally adopted in the treatment of threatened cases, or it may be through endeavours to relieve the overtaxed renal function by encouraging other channels of excretion as the skin and the bowels.

Other pathological phenomena, more or less frequent during pregnancy, affording evidence of defective metabolism and accumulation of toxins or of the toxin forming products include irregularities

of the skin, such as pruritis, pigmentation changes, etc., irregularities of the nervous system, such as depression of spirits, morbid cravings, tendency to melancholy, and headaches apart from the eclamptic form. (In epileptics we have an exaggeration of the disease,) irregularities of the vascular system, to which we shall refer, and irregularities of the urinary system such as glycosuria, acetonuria, etc.

(4) Secretion of milk and enlargement of the mammary glands must obviously still further add to the maternal work, and so tend to increase the strain on metabolism and accumulation of deleterious products.

(5) Increased cardiac area and other circulatory phenomena. Much current speculation prevails regarding the increased cardiac area. By some it is held to be due to hypertrophy of the left ventricle to meet the extra demands made upon the cardiac muscle in dealing with the increased volume of blood necessary for the foetal as well as the maternal requirements, also to deal with the tendency to increased blood pressure and increased metabolism. By others the increased cardiac area is due to the dilatation of the right side of the heart of which there is evidence in the pulsation of the veins in the neck. Others again hold that there is both dilatation of the right and hypertrophy of the left ventricle. According to Dr. Nicholson, who has

given this subject much careful consideration, the increased cardiac area is due to dilatation of the right side of the heart caused by overfilling and engorgement of the pulmonary vessels, a condition in turn due to excessive function of the suprarenal glands which, while causing constriction of the systematic arteries and arterioles, exercise a directly opposite effect in the case of the pulmonary veins.

The oedema and congestion of the lungs characteristic of many cases of pregnancy is due to this pulmonary venous dilatation and engorgement.

In addition to that of the pulmonary veins, dilatation of the veins of the neck has already been referred to. We also observe dilatation of the veins of the lower extremities occurring at a time before pressure of the uterus upon the pelvis can have so enlarged as to retard the returning blood therefrom to the heart. The cause ~~therefore is~~ here may be probably also due to overactivity of the suprarenal gland..

Such an abnormal condition of the state of the heart and circulation evidently adds to the impairment of the efficiency of the liver, especially in its urea forming function, and the general dilatation of the veins probably aids the tendency to thrombosis.

In the kidneys, excessive suprarenal function probably acts similarly by primarily effecting intense constriction of the renal arteries and

arterioles to such an extent that the blood supply to the minute vessels in the glomeruli is cut off and thus anuria and consequent retention of the urinary constituents in the system results. In this manner the blood becomes further laden with impurities which tend to increase the work of the organs of metabolism and is probably an important factor in bringing about autolysis especially of the liver. This renal condition will probably react on the heart and thus cause hypertrophy of the left ventricle in addition to the dilatation of the right side which we have already been considering. If this theory be correct, the suprarenal glands play a very important part in effecting the accumulation in the blood of the waste products at a comparatively early stage, and the enlargement of the thyroid gland which takes place later is evidence of an attempt on its part to counteract the evil influence of the former, attempts which are ultimately successful in those cases where Eclampsia does not occur, fortunately the majority of cases.

I have dwelt somewhat long on this fifth event in our series, but I trust that its important bearing on the subject of toxaemia and Eclampsia will afford sufficient excuse for its seeming tediousness.

(6) Enlargement of the thyroid gland, more or less characteristic of a large number of pregnancies, has already been dealt with, and as we have already

seen, must, in accordance with the latest theories of its function, play an important role in connection with the accumulation of toxins.

From consideration of the above series of phenomena have we not therefore good ground for concluding that, apart from a placental autolytic theory, there is sufficient material generated of a toxic nature to account for the occurrence of Eclampsia?

The causes of this toxicity, as we observed, are of a complicated nature, but are due in the main to partial or complete failure of the liver to cope with the ~~ammo~~ amino-acids, such failure again being due to autolysis arising from the excessive products of maternal and foetal katabolism with which the blood supplying the liver is laden, also as the outcome of renal incompetence on account of overactivity of the suprarenal glands during abeyance of the thyroid function.

The cause of the widespread thrombosis is liberation of nucleo-proteid from the disintegrated cells of other organs as well as the liver, aided probably by the venous dilatation.

The constant association of Eclampsia with high arterial pressure, thrombosis and accumulation of ~~ammo~~ Amino-acids and other nitrogenous products prima facie appears to justify the conclusion that one or all of these is closely connected with its causation

and from our consideration of the subject all appear to arise from certain pathological conditions occurring during pregnancy without the aid of placental elements having any influence in the matter. More especially does this appear to be the case when we take into consideration the occurrence of Eclampsia during the puerperium when no placenta is present. Page 5

Before proceeding further I would like here to remark that since entering upon the work of this thesis my attention has been directed to a series of very able and exhaustive articles on "Recent work on the Etiology of Eclampsia" by Dr. Eardley Hollands which appeared in the numbers of the "Journal of Obstetrics and Gynecology" for October, November and December last.

In the first of these articles Dr. Hollands remarks - "It is only by a close inquiry into the effects both anatomical and chemical produced on the body by Eclampsia that a conception of the true nature of the disease can be found. The clinical picture avails nothing here."

Now as I have drawn largely on the so-called "clinical picture" in the endeavour to establish the theory of the causation of Eclampsia which appears to me the most feasible, permit me to state that although doubtless as a result of post mortem examinations and also of chemical investigation, much knowledge

regarding the pathological condition of the various organs has been obtained and probably only by such a path will a solution of Eclampsia be reached, yet at the present time, notwithstanding all investigation, we are still in the dark regarding its true nature. Such being the case we are to a large extent compelled to fall back on clinical symptoms. Not only so, but our treatment is still to a large extent based upon such symptoms.

In support of his contention Dr. Hollands goes on to remark that other maladies which he considers of a different nature (and he is probably quite correct in so doing) have the same symptoms as Eclampsia. As an instance he adduces Uraemia and yet apparently is avowedly as much in the dark regarding the true nature of the one as of the other, in fact Dr. Hollands himself must base his view that the two maladies are essentially different upon the very "clinical picture" which he so lightly brushes aside, viz. chiefly that Eclampsia is associated with pyrexia, while on the other hand Uraemia is characterised by reduced temperature, also that convulsions are characteristic of Eclampsia, while their occurrence is rare in Uraemia.

Therefore I contend that the so-called "clinical picture" cannot thus be discarded as of no consequence and especially is this the case in general practice.

<u>To Sum up</u>, I would suggest that the direct cause

of Eclampsia is a toxin, or more probably a combination of toxins, either of a chemical or mechanical nature, or of both. That, on the one hand, these toxins are either amino-acids or their derivatives, or, on the other hand, are emboli, haemorrhages or aneurisms pressing mechanically on the brain substance. That the presence of amino-acids is due to failure of the liver to transform them into urea. That this failure is due to hepatic degeneration from autolysis, which in turn is caused by the blood supply becoming laden with waste products resulting from excessive maternal and from foetal katabolism as well as from retained urinary constituents on account of failure of renal elimination. That this latter is effected by overactivity of the suprarenal function in the temporary abeyance of that of the thyroid. That the emboli are due to the presence of nucleo-proteid liberated from the disintegrated maternal cells. That the haemorrhages and probable aneurisms are the result of the high arterial tension.

SYMPTOMS.

It is unnecessary to dwell long on the symptoms of Eclampsia, as they are quite unique and characteristic. They vary in accordance with the severity of the attack.

We are accustomed to such expressions as pre-eclamptic or prodromal and eclamptic symptoms, and for the purpose of treatment such a differentiation is very essential, not to say necessary, but when the pre-eclamptic symptoms appear, doubtless the real malady, from a pathological view, has reached a certain stage. Not only so, but from a study of the whole subject "Eclampsia" can only be regarded as the culminating climax or final manifestation of toxaemia of pregnancy. As, however, in the convulsive or so-called eclamptic stage new developments arise calling for special and more prompt treatment, the symptoms of these developments stand out distinct from the prior ones of toxaemia generally.

(1) Oedema, though not always present, is pretty general and is perhaps the first symptom to attract the attention of the superficial observer, especially when it occurs in primipara. This probably commences first in the lower limbs but is soon widespread over the whole body. In the more severe cases it resists pitting on pressure. It may make its appearance at various stages from the end of the sixth

month to the termination of pregnancy. This symptom is always ominous and should at once lead to examination of the urine, when it will almost invariably be found that albuminuria to a somewhat large degree has set in. The general practitioner, as a rule, has not the time to estimate the quantity of urea present, consequently a quantitative analysis is unusual, but we may conclude that the presence of albumin in the urine, especially to any great extent is an indication of diminished secretion of urea.

The oedema is accompanied by other symptoms as tendency to persistent vomiting which also precedes the oedema epigastric pain, tinnitus aurium, and especially almost invariably by severe headaches, at first with varying degrees of intensity and perhaps short intervals of cessation, but later with more persistence, until the headache becomes continuous. Such headaches are accompanied as a rule with dimness of vision. Ophthalmoscopic examination may reveal albuminuric retinitis which in Eclampsia is not considered by certain authorities to be of so serious import as in ordinary nephritis. Anuria, more or less complete, is always a marked symptom at this stage.

As we shall see when discussing the treatment this is the stage when prompt prophylactic measures are generally successful in warding off the more acute symptoms of the next stage.

Should the symptoms of this initial stage be

overlooked or neglected, sooner or later those of the second will make their appearance, and often when they do with startling suddenness. Such an untoward event may occur at any time during the latter months of pregnancy, as well as during labour and the puerperium; probably on rare occasions it may occur much earlier. One case is recorded as having occurred during the third month. It is doubtful, however, if this was a genuine Eclampsia.

The convulsions bear a close resemblance to those of ordinary epilepsy and probably may be of the same nature. It is held that eclamptic fits are peculiar in not having the initial cry of epilepsy, which may indicate that in the latter malady the muscular fibres controlling the movements of the vocal chords participate in the initial stages of the tonic spasm of the neighbouring muscles. The same probably occurs in Eclampsia, but as in pregnancy, especially at full term, and extending to a less degree into the puerperium, the lung capacity for breathing is limited, the air passing through the larynx and trachea is not on this account forced with the same energy or is deficient in volume to give rise to the audible cry characteristic of certain epileptic seizures.

It may therefore be that both in epilepsy and Eclampsia the immediate ~~central~~ cerebral cause is the same, the difference between the two being in the more remote or indirect cause, that of Eclampsia being due to some

special toxin peculiar to pregnancy which yet creates a cerebral condition common to both. As we have seen, in Eclampsia this condition may be caused by some form of mechanical pressure, and we know that certain tumours as well as pieces of bone pressing mechanically on the brain substance are often the exciting causes of epilepsy.

Reference has already been made to the greater predominance of Eclamptic cases occurring during labour ("intrapartum") evidently due to the uterine contractions acting as the exciting cause. The system, as we are aware, is at the time laden with toxins to such a degree as to supply the very conditions necessary for the liability to convulsions and requires only such an exciting cause to start them. The uterine contractions here act the part as it were of the spark which sets off the explosion in the mine where pent up gases of a highly inflammable nature exist. Consequently intrapartum Eclampsia is probably the most dangerous form and indicates the paramount necessity for immediate evacuation of the uterus, by such means putting a stop to the exciting cause. For the same reason perhaps post partum Eclampsia may be looked upon as the least dangerous kind.

Page 12

The convulsions are characterised by two distinct stages, first a tonic and then a longer clonic one. In the tonic the muscles of the face are first

affected; this condition rapidly extends downwards in a few seconds, involving the whole muscles of the body in a tonic spasm. Fortunately the duration of this stage lasts only for a few seconds, otherwise death of both mother and foetus would inevitably accrue from asphyxia. This is followed by a clonic stage lasting for a few moments during which the muscular spasms are of a rythmical character, i.e. violently contracting and relaxing alternately and giving rise in the face to hideous contortions. During both tonic and clonic stages the patient is unconscious and may void both urine and faeces involuntarily. A condition of coma follows the convulsion which, if the fit is a solitary one, gradually passes off and the patient, at first somnolent, regains consciousness. The occurrence of only one fit, however, is exceptional. We are more likely to meet with a succession of fits rapidly following one another, varying from two or three up to any number. Cases have been recorded of over 100 fits. Should the fits thus succeed one another in rapid succession, the intervening coma is complete without a return to consciousness during the whole period of their duration. The greater the number of fits, the more grave is the prognosis. Should the number exceed 20, it becomes very grave. Yet in such desperate cases hope should not be abandoned nor curative measures given up till every known resource has been exhausted. An incentive to persevere to the

last is afforded by the success which has ultimately crowned the persevering efforts of obstetricians in certain cases apparently hopeless, notably that of Sir Halliday Croom in 1909 recorded in the Edinburgh Medical Journal, where after the occurrence of a very prolonged succession of convulsions his treatment eventually by decapsulation of the kidney was crowned with success.

Severe cases of Eclampsia are often followed by serious consequences of varying periods of duration, e.g. partial or total loss of memory of events which took place for a longer or shorter period before the commencement of the attack. In one of my cases, viz: that of Mrs J - No. 7 loss of memory of this nature for about two or three months even after she was able to go about and attend to her duties was a marked feature. Other grave sequelae may occur, such as blindness of varying degrees, and even insanity, which latter may be permanent. Such sequelae, however, are comparatively rare.

TREATMENT.

Our endeavours at treatment should be directed -

(1) To lessen the amount of toxins circulating in the blood.

(2) To counteract the effects of the toxins.

(3) To soothe the nervous irritability and control as far as possible the convulsions by drugs whose beneficial effects in this direction are well known.

(4) To lower the high degree of blood pressure and counteract the tendency to coagulation of blood.

(5) To terminate the labour in certain cases where this is advisable.

(6) To adopt special surgical treatment in certain extreme cases.

In pursuance of the above we are according to circumstances to adopt prophylactic measures in the prodromal stage and more active treatment where the convulsions have already occurred. Although there is a presumption that special types of Eclampsia, having separate origins and occurring at different stages, may exist, yet as this is far from being settled we cannot in the circumstances vary our treatment to meet those supposed different types.

If we are fortunate enough to detect the patient

in the prodromal stage of anuria and albuminuria, absolute rest in bed should be enjoined and a sharp purge of calomel and jalap should be at once administered and free purgation thereby procured with a view of (1) emptying the intestine and thus cutting off at its source the supply of products which feed the blood with toxins; (2) to vicariously relieve the strain upon the overwrought kidney by procuring a watery flow from the bowels instead. It is to be borne in mind that, in addition to the ~~choloque~~ cholagogue cathartic and antiseptic properties of calomel, it has also a diuretic effect which almost invariably follows its administration in such cases as we are considering. The dose of calomel in order to be effective must be a large one. Dr. Reynolds Watson advocates the administration in such cases of a dose of 10 grains to be repeated in five or six hours if necessary. The result is usually an abundant polyuria. Thyroid gland extract in 5 grain doses thrice daily should be administered and continued up to the termination of labour even after the disappearance of the albuminuria. This, as we have seen, tends to favour the return of normal metabolism and of diuresis, probably by its vaso-dilatory function. In addition diuresis should be encouraged by large doses of acetate of potash, say 30 or 40 grains thrice daily. It is most important that the diet should be restricted to milk and to milk alone, say at least 12 tumblerfuls per diem so long as

any trace of albuminuria exists. The bowels must be kept in a laxative state up till the termination of labour.

By the adoption of such measures we will almost invariably be fortunate enough to arrest the further progress of the disease at this stage even in cases where the symptoms when first discovered have so far advanced as to be alarming, foreshadowing the almost immediate onset of convulsions. I refer to such cases where the albuminuria is very pronounced, and the albuminous deposit almost becoming solid on boiling. Having succeeded in arresting the albuminuria, the patient should be kept under strict surveillance until the termination of labour. Should there be the slightest tendency to any increase of the albuminuria, resort must again be had to the large dose of calomel as at first and thus keep the symptoms within the safety zone. It is probable that in most cases where the albuminuria persists, there has been pre-existing renal disease.

From the above considerations, the great importance of examination of the urine in all pregnant women, especially in primipara, is very evident. This should be done at stated intervals, say fortnightly from the sixth month on to the termination of labour. As such precautionary measures are now probably universally adopted by the new generation of general practitioners, statistics in all likeli-

hood will in the near future indicate a much lower percentage of eclamptic cases and consequently a lower mortality in labour cases generally. I am unaware whether such statistics have as yet been obtained, but from my own limited experience since adopting this practice, I have not had a case of Eclampsia, i.e. the convulsive stage, for a number of years.

Should, however, we miss the opportunity of treating the prodromal symptoms, and suddenly be confronted with the convulsive stage, before proceeding to empty the uterus at once as advocated by some, I am of opinion that we should still continue, although now in a more prompt and heroic manner, our endeavours to eliminate or counteract the poison and to encourage diuresis. For the former purpose, two channels are especially at our disposal, viz: the skin and bowels. To utilise the former we should, when no contra-indication exists, administer $\frac{1}{10}$ to $\frac{1}{8}$ of a grain of pilocarpin. Personally, from my experience of this drug in other cases as well as in Eclampsia, I do not share the apprehensions of some as to its supposed deleterious effects in every instance on the heart and circulation. Apart from its diaphoretic effect, it should exercise a favourable influence on the high arterial tension of Eclampsia. Its administration is followed in a few minutes by profuse perspiration. Simultaneously

with the pilocarpin, in order to soothe the nervous excitability, control the convulsions, and also aid diaphoresis, ½ grain of morphia should also be administered hypodermically. The morphia should also tend to lower the high blood pressure and probably also encourage diuresis. Thyroid gland extract, either hypodermically or per rectum, should in addition be administered about the same time in large doses from 20 to 30 grains. The rationale of this has already been considered. To still further encourage diaphoresis the patient should be surrounded by a number of hot water bottles placed in contact with the skin of the feet, legs, thighs and under both axillas, precaution being taken to avoid blistering of the skin. Hot packs and the steam apparatus have been highly recommended for the same purpose and in hospitals where such expedients are always at hand, this latter is the more effectual mode of procedure, but in general practice, especially amongst the poorer classes, where the practitioner has to rely upon the most prompt measures at his disposal, the case being one of extreme urgency, the simpler and quicker method of the use of hot bottles as described above should rather be adopted as valuable time may be lost before the hot pack or any steaming apparatus however simple can be got ready, and probably the hot bottles are after all as efficacious as the other methods. Ordinary quart or pint bottles can easily

be obtained and these serve the purpose admirably. During the above procedure chloroform inhalation should be cautiously administered. In the exceptional circumstances this may be left in the hands of an attendant or nurse, the physician meanwhile, as far as possible, keeping an occasional watch over its administration. In one of my cases, chloroform probably to a large extent aided in stopping the convulsions. A soap and water enema should immediately follow the above measures as well as washing out of the stomach. By such methods in a considerable number of cases diuresis will be established and the convulsions will be arrested or rendered much less severe and less frequent, and in such cases subsequent treatment should of course be the same as that adopted in the prodromal stage. An instance of the successful results of such treatment is afforded in the case of Mrs Glendinning No. 3 who, after the occurrence of 5 fits about the seventh month of pregnancy was safely tided over till near the full term when she was safely delivered of a living foetus and made a good recovery, the albuminuria ultimately entirely disappearing.

Should the convulsions continue in spite of the above measures, the following treatment should be adopted:- (1st) The adoption of venesection and by this means relieving the high arterial tension, as well as ridding the system of part of the toxin

laden blood; (2nd) the subcutaneous injection of saline solution containing acetate of potash or acetate of sodium in addition to the sodium chloride. Dr. Jardine of Glasgow who first drew attention to the importance of this mode of treatment, considers that normal saline solution without sodium acetate is devoid of diuretic action. He, however, considers the potash salt as poisonous, but others hold the opinion that acetate of potash is quite safe and more effectual than the sodium salt. The injection of the saline solution replaces the poisoned blood which has just been removed through the venesection by a pure fluid free from toxins. The increased volume of fluid in the vessels will also probably tend to dilute the constituents of the blood and thus favour diuresis and renal elimination as well as wash away the deleterious products blocking up the tubules; (3rd) washing out the stomach and administering copious enemata and thus preventing further intoxication of the blood from the digestive tract; (4th) the administration of an enema of 30 grains of chloral hydrate and 30 grains of potassium bromide. Veratrum viride, a remedy which has been more used in America than in this country for its effects in lowering arterial tension and slowing the pulse, has been strongly recommended by Prof. Stevenson of Aberdeen, who has had remarkably good results from its judicious use. Prof. Mangiagalli of Milan, who

is also a strong believer in its efficacy, considers it, not as an antidote for Eclampsia, but only as a "remedy for its most violent manifestations, the convulsive fits". It would appear to be worthy of a more extensive trial than hitherto adopted here.

We are indebted to Dr. Justin McCarthy for bringing into prominence nitroglycerine administered hypodermically, a remedy which is often followed by very favourable results in cases of Eclampsia. Its effects appear to be somewhat similar to that of veratrum veride but probably much safer to use. Its administration should also be adopted in the class of cases we have just been considering.

Dr. Emgetmam and C. Stade recommend Leech Extract (Hirudin) and state that, if injected in time, it will cause cessation of the convulsions by inhibiting the tendency to coagulation of the blood.

Should failure to arrest the disease still continue in spite of all methods hitherto adopted, emptying the uterus now becomes imperative. The adoption of such procedure would seem to be strongly suggested by the circumstance that in certain cases death of the foetus in utero or its delivery tends to arrest the convulsions and otherwise effects a re-establishment of the normal functions. Such a circumstance would almost suggest the idea of artificially procuring the death of the foetus in utero were we absolutely certain that such an extreme procedure

would be followed by cessation of the convulsions, especially when we consider that, in all probability, the life of the child will be forfeited in any case; and if, in other circumstances, in order to save the life of the mother, craniotomy is considered justifiable, the same reasons might with greater force be adducible here.

Having determined to empty the uterus, we will now consider the safest and speediest methods of accomplishing this. At a comparatively early stage, when the os is not only rigidly closed, but when the cervix is not wholly taken up, manual dilatation is out of the question; also attempts at dilatation by metallic dilators, such as Bossi's, are difficult and attended with the risk of serious laceration of the cervix and consequent risk of septicaemia. In the circumstances we have to rely on what is more properly surgical procedure, and perhaps at this stage, when the vaginal canal is comparatively narrow, vaginal Caesarean Section preceded by episiotomy is the safest and quickest method to adopt.

At a later stage, when the cervix is wholly taken up and disappears, manual dilatation may now be attempted; failing this, the use of metallic dilators, such as Bossi's, is now justifiable, being more expeditious than Champetiers de Ribes bags. The use of such metallic dilators certainly lies within the province of the general practitioner.

At a still later stage, when labour has actually commenced and the os is undergoing normal dilatation, this should be aided manually and the labour terminated as expeditiously as possible by the aid of forceps or otherwise as the presentation admits.

Should the convulsions continue into the puerperium, or should their first appearance then take place and failure still follow the adoption of the above treatment, two other surgical methods recently inaugurated remain to be tried. These are Lumbar Puncture and Renal Decapsulation.

Lumbar Puncture is founded on the idea that increased cerebro-spinal tension exists in Eclampsia, which is reduced by drawing off fluid from the spine. It is stated that in those cases where a free escape of fluid occurred after puncture, a good result followed. On the other hand, where the escaping fluid ~~is~~ was very scanty or only in drops, the reverse ~~is~~ was the case. It is thought that the good results of lumbar puncture are only probably of a temporary nature, the fits recurring after a time. If so, it can therefore only be palliative, but this allows time to be gained and opportunity afforded for preparation for the other and apparently last resource, viz: Renal Decapsulation. In a number of cases, however, it has been followed by success without renal decapsulation having to be resorted to afterwards. One case recorded by Dr. Jardine of Glasgow

having recovered by this means after the occurrence of 200 fits.

What would appear to be a last resource, so far as our present knowledge extends, is Renal Decapsulation. It was first tried in ordinary nephritis, but appears to have been abandoned on account of its unsatisfactory results. It was first introduced into obstetric practice by Edebohls, and although far from being universally successful, the number of cases of recovery following its use justifies its adoption in these desperate cases where all other methods have failed. This was well illustrated in the case of Eclampsia of Sir Halliday Croom's already referred to, where, after the other important methods of treatment had failed, renal decapsulation as a last resort was accomplished with complete success and recovery of the patient.

The beneficial results of this operation would appear to be due to relief of tension of the renal circulation allowing blood to gain entrance more freely into the vessels of the glomeruli and thereby bringing about diuresis. Wet cupping of the back in the region of one or both kidneys might indirectly have a similar result, and I would suggest that such should be tried before proceeding to the more serious operation of renal decapsulation. I am encouraged to make such a suggestion from my own experience in the case of Mrs J., one of the cases of Eclampsia

which fell to my lot, when, after exhausting all other methods which we could think of without success, the convulsions continuing after emptying the uterus, I applied wet cupping to the region over one of the kidneys, and had the satisfaction of seeing the disease arrested. Not one convulsion occurred after this operation, the patient ultimately making a good recovery.

SHORT SUMMARY OF TREATMENT.

I. Object of Treatment.

(1) To minimise entrance of toxins.

(2) To counteract toxin.

(3) To control nervous excitability.

(4) To lower blood pressure.

(5) To combat tendency to coagulation.

(6) In certain circumstances to empty the uterus.

(7) In extreme cases to arrest disease by the surgical measures,- Lumbar Puncture and Renal Decapsulation.

Prodromal Stage :

(1) Rest in bed.

(2) Exclusive milk diet.

(3) Purgation by calomel assisted by other drugs.

(4) Thyroid gland administration.

Convulsive Stage :

(I.) Where immediate evacuation of uterus not desirable or necessary.

(II.) Where immediate evacuation of uterus is imperative.

(I.) Where not desirable :

(1) Same as in prodromal stage with increase of thyroid.

(2) Diaphoresis:-

(a) Medicinally.
(b) Mechanically.
(c) Mechanical evacuation of stomach and bowels.

Convulsive Stage (Contd.)

(I.) Where not desirable (Contd.)

(2) Diaphoresis.

(a) Medicinally - hypodermic administration of pilocarpin when advisable, assisted by hypodermic administration of morphia.

(b) Artificially - e.g. hot bottles, hot pack or steam.

(3) Overcome nervous excitability, e.g. morphia injection ½ grain, sometimes by chloroform.

(4) Encourage metabolism and favour elimination of toxin.

(a) Thyroid gland extract.
(b) Wash out stomach and administer enema.

Convulsive Stage still persisting :

(1) Intravenous injection of saline solution.

(2) Venesection.

(3) Enema of chloral and bromide.

(4) Nitroglycerine.

(5) Veratrum veride.

(6) Hirudin.

(II.) When immediate evacuation of uterus imperative.

(a) Early stage before cervix taken up.

(b) Later stage after disappearance of cervix.

(a) Early Stage

(1) Episiotomy.

(2) Vaginal Caesarean Section.

(3) As in ordinary labour but accelerated.

(b) Later Stage

(1) Try manual dilatation of os.

(b) Later Stage (Contd.)

(2) Metallic dilatation, e.g. Bossi.

(3) Deliver as in ordinary labour by forceps, turning, etc.

When labour has already commenced.

Same as above.

(III.) Puerperium Eclampsia after failure of other methods.

(1) Wet cupping.

(2) Lumbar Puncture.

(3) Renal Decapsulation.

CASES OF ECLAMPSIA OCCURRING IN MY PRACTICE.

Case No. 1

Mrs O. Primapara. Age 37.

Membranes ruptured December 15th 1887. Shortly afterwards labour pains of short duration and occurring at long intervals commenced. I was summoned to attend her on the afternoon of the 16th December. This would be about the full term of pregnancy. I found very pronounced oedema all over the body. She complained of severe headache and occasional attacks of blindness. The urine was found to be loaded with albumen. On examination the os was found to be closed and rigid. The pains were of a very painful and severe nature. I left her to allow os to dilate and returned at 8 p.m. The os was then dilating very slowly and still very rigid. I again left her and returned about 10 p.m. and remained with her all night. About 3 p.m. the os would probably be dilated to the extent of the size of half a crown, the pains now following each other at short intervals probably of about 5 minutes. Suddenly about this time she complained of an attack of blindness and was seized with a characteristic Eclamptic fit. The person who was acting as nurse being untrained for that capacity, took fright and ran out of the room

leaving me alone with the patient who soon recovered from this fit but remained comatose. In a few minutes a second convulsion followed. I then administered choloroform which seemed to greatly mitigate the severity of the convulsion. The os was then almost fully dilated and shortly afterwards I was enabled to apply the forceps with ease, the presentation being a normal head one. No more fits occurred and delivery was terminated at 5 a.m. 17th December 1887, a female child being born alive. The albuminuria gradually decreased with returning diruesis and in about three weeks or so afterwards the urine was free from albumen. The patient made a very slow recovery and was unable to rise from bed for about six weeks afterwards. During this period her temperature continued high, about 102°. There was probably, however, a certain degree of Septicaemia which might account for the slow recovery and high temperature.

This patient did not conceive again but is still alive, now more then 22 years after the event and in spite of a slight cerebral haemorrhage about a year ago, she is comparatively well and able to attend to her ordinary duties.

The noteworthy features of this case are the age of the patient, the prolonged and exhausting labour, the occurrence of the Eclampsia in the second stage and the apparently mitigating effect of the Chloroform Seeing the case afforded the prospect of a speedy

delivery and considering that my efforts were hampered for lack of proper nursing assistance, I did not wait to try the effect of either morphia or pilocarpin administration but endeavoured to hasten delivery as quickly as possible.

Case No. 2.

I regret that, as this case was ushered upon me so suddenly and without any warning, I had no opportunity to take notes, the time having to be wholly taken up in active measures to endeavour to relieve the symptoms. The following account is therefore from memory, consequently such details as dates may not be absolutely correct, otherwise the outstanding events occurred as I am about to relate them.

Mrs S. Primapara. Age about 25 years.

The date of the occurrence would be about 1889.

I was summoned first to attend her in the evening (say) April 5th. I found the os just beginning to dilate and rigid. Pains were very severe following each other at short intervals and patient appeared to be much exhausted, more so than she should have been at this stage. I remained with her for several hours. At the end of this time the dilatation had made little progress, the os having opened to probably not more than the size of a shilling and still rigid. There was no oedema nor outward symptoms to indicate the near onset of Eclampsia.

About 6 a.m. I left for a short interval, intending after a time to return when I considered that dilatation had more fully developed. About 8 a.m. I was summoned hurriedly to attend her, being informed that she was in a fit. On arriving at her

house I found her in a violent convulsion. Dr. Ritchie, happening at the time to be in the neighbourhood, kindly responded to the urgent request for assistance. On inspection of the urine it was found to apparently contain a large proportion of blood. About $\frac{1}{10}$ grain of pilocarpin was then administered, but, although diaphoresis followed, the fits continued in close succession without almost any interval. On examining the os I now found it comparatively soft and almost fully dilated. Dr. Ritchie then kindly administered chloroform and I managed with comparative ease to fully dilate the os manually and to apply forceps and in a short time to deliver a living child. The fits, however, still continued in the same persistent manner even after expulsion of the placenta which was effected in a few minutes after delivery of the foetus. Dr. Ritchie now reluctantly had to leave me to attend to an urgent case of his own, and being deprived of his valuable assistance and somewhat forlorn encouragement, I sent for Dr. Barbour who very kindly and promptly responded to my call. He advised me rather to encourage subinvolution of the uterus than otherwise so as to allow postpartum haemorrhage within certain bounds to remain unchecked in the hope that this might indirectly encourage renal elimination; but all was of no avail, the patient dying in about one hour after delivery in a convulsion.

The outstanding features here were the tediousness of the labour and the association of the exceptional persistence and severity of the convulsions with the blood in the urine, probably indicating the association of uraemia with the Eclampsia, finally the fact of the Eclampsia occurring intrapartum.

Two questions naturally suggest themselves:-
(1) Would this patient have had a better chance of recovery had morphia or thyroid gland extract been administered?

Of course this occurred more than twenty years ago, at a time when morphia administration in such cases was to a large extent regarded with suspicion, its beneficial effects being then only realised by a few. The severity of the case, however, with the haematuria indicating the additional presence of acute nephritis, would almost seem to preclude the possibility of a favourable issue from morphia administration or from that of thyroid gland. Otherwise the rapidity of the case terminating in death afforded no opportunities for attempting further modern remedies, even although we had been aware of them at the time. Probably a period of not more than two hours would elapse from the commencement of the onset of convulsions till death closed the scene. The fits followed each other in such rapid succession that the patient almost appeared to be undergoing

one continuous convulsion all the time.

(2) The second question which suggests itself is,- Had prophylactic measures been adopted at an earlier stage when albuminuria was beginning to develop, would she have had a chance of recovery?

The solution of this query depends upon whether acute nephritis actually existed along with the Eclampsia or not. If it did, it is extremely doubtful if prophylactic measures would have warded off the attack. On the other hand, if the Eclampsia existed alone, in all probability a successful issue would have resulted.

Case No. 3.

Mrs G. Age 27 years. Primipara 1893.

March 24th. Had by this time reached about the seventh month of pregnancy. General oedema over whole body. Pitting of skin on pressure. Severe headaches. Urine scanty and found to be loaded with albumen.

March 28th. Was summoned suddenly to attend her and found her in a convulsion which was followed by a short period of coma from which she recovered to partial consciousness. This was soon followed, however, by a second fit. $\frac{1}{10}$ - $\frac{1}{8}$ grain of pilocarpin was injected and I sent immediately to Dr. Haultain for assistance who very promptly responded to my call. By this time the convulsions were succeeding one another at short intervals, coma completely intervening without any return to consciousness. By the time of Dr. Haultain's arrival she would be in the fifth convulsion. He immediately injected $\frac{1}{2}$ grain of morphia hypodermically and ordered the trunk and limbs to be surrounded with hot water bottles and the patient covered with blankets. This was almost immediately followed by profuse perspiration, after which there were no more convulsions. The urine drawn off was found to be loaded with albumen almost solid. After the last convulsion patient continued for about one hour apparently comatose. This was

followed by profound slumber, and in about three or four hours she regained consciousness, at first only partially, but ultimately completely.

She was then put on an exclusively milk diet with in addition effervescing potash or soda and ordered to take a mixture containing acetate of potash in doses of 30 grains thrice daily. Free evacuation of the bowels by purgatives was enjoined.

April 12th. Oedema gradually disappearing. Albuminuria slowly decreasing and diuresis becoming daily more abundant.

From 13th April on till May 20th, when she was delivered of a living child, the proportion of albumen in the urine continued much about the same, only a slight cloudiness in the urine becoming apparent after boiling with nitric acid. There was nothing unusual about the labour, this being effected by the aid of chloroform and forceps. The foetus when born was very thin and badly nourished and did not survive longer than a few weeks. Albuminuria continued in much the same proportion for about three weeks after labour and ultimately completely disappeared, and the patient made a good recovery.

She again became pregnant in about six months, and having by this time removed to Leith, I did not attend her during her next labour, but had a visit from her about two months before this event took place, she having become alarmed at seeing a deposit

of urates in her urine and imagining that such a condition was a presage of another attack of Eclampsia similar to what she had experienced during her first pregnancy. I however found the urine to be entirely free from albumen, and her confinement took place at full term unattended by Eclampsia and otherwise normally.

I have learned that since then she has had three or four confinements safely without complications, with the exception that, after her latest, she developed some form of mental derangement or insanity, and has been under treatment in an asylum for a number of years where she is to-day, her mental condition being looked upon as hopeless. This unfortunate state of matters, however, has probably no connection with the Eclampsia of her first confinement.

Such a case of Eclampsia as this is not an uncommon type of the malady, and the fact of convulsions having commenced might at first sight indicate a condition for the immediate evacuation of the uterus. However, the success which followed the measures adopted here should rather encourage us first to endeavour to favour elimination of the pent up secretions with their toxins by other channels than the blocked up kidneys, such as the skin and bowels, at the same time checking the tendency to convulsions by such valuable remedies as morphia. A special feature of this case is the convulsions occurring

antipartum.

A feature of such cases, where the patient is restricted to milk diet for a considerable time, is the puny and ill-nourished condition of the foetus if born alive. This may indicate that the now favourable maternal condition may to some extent be aided by decreased metabolic activity in the foetus, so that its waste products are thus minimised and not only more easily eliminated by the maternal organs, but the quota of toxin producing material from the foetal source is thereby much lessened, relieving the maternal metabolism to this extent, the reverse of this being seen in more serious cases where we invariably have a fully developed foetus.

Case No. 4.

Mrs R. Age 27 years. Primipara.

March 13th, 1901. Summoned to attend her about 4 p.m. Found her in convulsions. (She would by this time be about 30 days from the full term of pregnancy.)

Previously during the day she had complained of severe headache with dimness of vision, sickness. I found marked oedema all over the body and had been informed that this condition had been gradually developing for the past six weeks or more; anuria almost complete.

I sent immediately for Dr. Haultain and meanwhile injected about $\frac{1}{6}$ grain pilocarpin subcutaneously and surrounded legs, thighs and trunk with hot water bottles. Soon profuse diaphoresis followed. Dr. Haultain, who again very kindly promptly came to my assistance, on arriving about 6 p.m. immediately injected $\frac{1}{2}$ grain morphia hypodermically. The convulsions, however, still continued to succeed one another with unabated severity, complete coma intervening.

9 p.m. Administered $\frac{1}{4}$ grain morphia.

12 midnight. Fits had now numbered about 15; another $\frac{1}{4}$ grain morphia administered, after which fits ceased, but coma continued without intermission all night. Urine drawn off by catheter found to be almost solid on testing with albumen.

March 14th. Still comatose. Urine still

loaded with albumen.

March 15th. Still coma. Urine in same condition.

March 16th. Signs of regaining consciousness; slight decrease of albumen.

March 17th. Still to a certain extent comatose. Albuminuria much the same as on previous day.

March 18th. Still slightly comatose but improving; able to swallow a little milk. Albuminuria still profuse.

March 19th. Gradually regaining consciousness; somnolent. Albuminuria apparently in much the same condition as previous day.

March 20th. Only partially conscious; still considerable albumen in urine. About 8 p.m. labour pains began. Os rigid and dilatation very slow.

March 21st. Os gradually dilating during the day. About 7 p.m. os almost fully dilated. Completed dilatation manually, then administered chloroform and delivered dead foetus with forceps. Presentation was a head one. Delivery over about 9 p.m.

March 22nd. Still semi-conscious. Commencing diuresis. Albumen in urine considerably lessened. Oedema gradually disappearing.

During above period daily evacuation of bowels by enemas.

March 23rd. Consciousness completely regained. Strictly milk diet; evacuation of bowels. Oedema

fast disappearing. Albumen in urine less than previous day.

From this time onward till about the 14th day of the puerperium the albuminuria was gradually disappearing and about that time ceased altogether, diuresis becoming superabundant. She made a slow recovery and about twelve weeks elapsed before this was complete.

November 1901. Became again pregnant and progressed favourably till May 1902 when she went for a short holiday to Millerhill. There being no w.c. in the house at Millerhill, she rose about 2 a.m. or 3 a.m. one morning and went out to a privy in the garden to evacuate the bowels. While in the privy she was seized with either a convulsion fit or an attack of syncope and lay there for about four hours, no one in the house being then aware of the occurrence. On regaining consciousness she returned to bed. Beyond a slight degree of shock, however, no evil effects apparently followed. On her return to Edinburgh she brought me a sample of urine, which I found on examination to contain a small trace of albumen. I then administered calomel and jalap, put her on a strictly milk diet, and kept her for a few days in bed, at the same time administering a diuretic of acetate of potash. The albumen entirely disappeared. Labour terminated safely at full term with the delivery of a living child, and she made a

good recovery.

Since then she has had two pregnancies with labours at full term and living foetuses without the occurrence of albuminuria.

In this case as in the last, the importance of first endeavouring to mitigate the severity of the symptoms before proceeding to empty the uterus is fully vindicated. It is also noteworthy that the death of the foetus, which in all probability occurred a day or two before delivery, did not bring about a cessation of the symptoms, at least of the coma, although it might be the case that the cessation of the convulsions took place after the foetal death.

When the foetus was born it was fully developed and had the appearance of a full time child, which circumstance may have aided the severity of the convulsions in the manner I have indicated in the previous case.

Case No. 5.

The following case illustrates the prodromal stage of Eclampsia and its treatment.

Mrs C. Age 24 years. Primipara.

Arrived in this country from her home in America in order to be confined at her father's house about end of October 1909.

September 10th. Engaged my services for her coming confinement, which was expected in about from 14-21 days. I then noticed that she was markedly oedematous, and on enquiry elicited the fact that she suffered from severe headaches and that the urine passed was scanty. Suspecting albuminuria, I at once ordered her to bed, enjoined a strictly milk diet and prescribed calomel grs. 5, to be followed by a strong Seidlitz. The urine, as I suspected, was found to be loaded with albumen almost "solid", indicating the supreme risk of the near onset of convulsions.

September 10th - 12th. Condition remains almost unaltered.

September 12th. Albumen less but still considerable. Oedema still present; diuresis established but not to any great extent. I then put her on thyroid gland extract 5 grains thrice daily.

September 14th. Slight improvement in all the symptoms.

September 15th. Headaches almost gone.

Albuminuria much decreased and diuresis fairly well established. Labour pains commence early in the day. Os dilates easily. About 5.30 p.m. administer chloroform and deliver with forceps with comparative ease. Labour over about 6.30 p.m. Foetus fully developed and alive.

September 16th and following days. Puerperium normal; albuminuria gradually disappearing.

September 28th. No trace of albumen. Patient able to rise about fourteenth day after delivery.

This case illustrates the supreme importance of examination of the urine, especially in primipara during the later months of pregnancy. From all indications she was doubtless at the very threshold of the convulsive stage, and for certain would have entered it had not her imminent danger been observed and measures adopted just at the last moment to arrest any further progress of the mischief.

Case No. 6.

Mrs R. Age 24 years. Multipara. Second pregnancy; first occurred about two years previously without Eclampsia.

June 1902. In answer to urgent summons, arrived at house about 5 p.m. and found her in a convulsion fit. Was informed that on one or two occasions during the previous month she had suddenly fallen down unconscious when attending to her ordinary duties, this however being looked upon as an ordinary fainting fit and made light of. There was marked general oedema, and before my arrival there had been two or three fits, so I was informed. After a hypodermic injection of ¼ grain morphia, I got her body and limbs surrounded by hot water bottles. This was shortly followed by profuse perspiration. The os being now almost fully dilated, chloroform anaesthesia was induced and delivery easily effected about 7 p.m. by the aid of forceps. No more convulsions followed. She was however in a semi-comatose condition for about one hour afterwards. During the first three or four days of the puerperium the oedema gradually subsided. The urine, which was at first loaded with albumen, gradually became free from this product, and in about fourteen days there was little or no trace left. She made a good recovery without any further complications and was able to get about in about fourteen days after the labour.

For the above description I have had to trust to memory, as I did not take notes of the case, consequently small details such as the days of the month may not be absolutely correct. The outstanding features, however, viz: the occurrence of the previous so called fainting fits, the oedema, the convulsions, and the normal recovery took place as related.

Case No. 7.

Mrs J. Age 42 years. Multipara. Had had nine previous confinements without serious complications and in addition one miscarriage, about fourth month of pregnancy. For about fourteen days prior to occurrence of Eclampsia had been nursing one of her children who was suffering from appendicitis. This entailed almost constant strain night and day and during this period she must have had little sleep.

September 6th, 1902. About 10 a.m. seized with violent headache. About 6 p.m. pains in her back began, occurring at regular intervals. At this time she would be fully more than eight months pregnant. Under the impression that labour was commencing, I was summoned and arrived about 7 p.m. On examination I found the os closed although soft and the cervix only partially taken up. Under the impression that the pains were false, I ordered her to have 1 grain of opium in pill form orally. I then left, but was summoned again about 10.30 p.m. to attend her in a convulsion. This was soon followed by a second followed by coma without any return to consciousness. $\frac{1}{10} - \frac{1}{8}$ grain pilocarpin was injected and the body and legs surrounded by hot bottles. Profuse diaphoresis followed shortly. I then sent for Dr. Frost who very promptly came to my aid. $\frac{1}{2}$ Grain of morphia

was now injected, but the convulsions continued with apparently increasing severity, with only short intervals of complete coma between. The convulsions at first began with twitching of eyelids and the mouth was drawn to left side. There appeared to be little or none of a tonic stage, but clonic spasms, commencing first in muscles of face, extended quickly over region of muscles of arms, body and legs. The fit would last about one and a half minutes. After fit pupils somewhat small did not react to light. Pulse about 110, increased tension, and temperature 102°. Respirationsvery much quicker than normal. There was no oedema. In spite of profuse perspiration there was no abatement of the convulsions. We then determined to empty the uterus. After the vagina had been rendered as aseptic as possible by a Lysol douche, I administered chloroform and Dr. Frost proceeded to dilate the os with a Bossis dilator. This was carried out with extreme care and about fully half an hour elapsed before the os was fully dilated. I may here mention that this was the first or second occasion on which this instrument had then been used in Edinburgh. The head was then found to be above the brim of the pelvis. Dr. Frost then applied the forceps and in about fully another half hour succeeded in delivering the head occipito posteriorly. In about ten minutes afterwards the placenta was delivered. There was not much postpartum haemorrhage.

I have no recollection of the occurrence of any convulsions during the delivery. Delivery was over about 1.30 a.m.

5 a.m. a convulsion, 7.30 a.m. convulsion, both of a severe nature.

During all this period, from the time I called about 10.30 p.m. on the previous evening, she had not regained consciousness.

September 7th, about 9.30 a.m., hot pack used followed by profuse perspiration. Bladder emptied by catheter, about 6 ounces of urine being the amount. Dr. Frost got a quantitative analysis taken and it was found to contain 3 grms. of albumin to the litre, some blood granules, and blood casts; urea 1.2%.

About half an hour after the application of the wet pack another convulsion took place. About noon two convulsions occurred following each other with only a short interval between.

Other convulsions occurred at 12.45, 2.45 and about 3 p.m.

4 p.m. Soap and water enema and patient surrounded again with hot bottles. There was a cessation of the convulsions till about 6.45, when four followed each other in rapid succession, each one more or less violent than formerly and of short duration.

About 9 p.m. when Dr. Frost called, the temperature was 104.2 and pulse 120.

11.15 p.m. Temperature 101.2 and pulse 106.

Wet cupping was about this time applied to region over one of the kidneys. No further convulsions.

September 8th. Temperature about 99°, pulse 86. Urine drawn off by catheter again; examined quantitatively by Dr. Frost was found to contain 0.75 grm. albumin to the litre. Urea 2.5%.

In the evening patient showed slight signs of returning consciousness, e.g. opened eyes when spoken to.

September 9th. Temperature 98.4, pulse 106. Could then swallow a small quantity of milk. Urine again examined by Dr. Frost contained 0.5 grm. albumin to the litre.

Later on during the day patient attempted to speak incoherently.

September 10th. Patient now fairly conscious but had no recollection of events occurring for some time prior to the Eclampsia. Temperature normal, pulse about 100; well marked jaundice.

Subsequently the patient gradually improved. The albumin in urine gradually diminished. Jaundice disappeared in about 4 days. Diuresis soon became well established and in about 14 days there was only a small trace of albumin which ultimately disappeared altogether. In about four weeks she was able to rise from bed. During the first part of this period her diet was solely milk, later when there was only a trace of albumin, she was put on farinaceous food,

and later still on white flesh. For about 2 months afterwards, however, the loss of memory continued. The child which was born alive was somewhat thin and badly nourished and indicated about 8 months of utero-gestation life. It gradually put on flesh, however, and appeared to be developing normally till a few months after birth when it died from an attack of Bronchitis

Mrs J. is alive and well at the present day.

The chief features of this case are (1) the age of the patient, (2) the fact of her being a multipara, (3) the prolonged and exhausting strain entailed by nursing her child preceding the attack of Eclampsia, (4) the cessation of the fits after the application of wet cupping, although it is to be noted that the fits were becoming less in severity before this was tried. Reference has already been made to the probable beneficial effects of wet cupping under the discussion of Treatment.

Case No. 8.

Mrs W. Multipara. 26½ years old. Had one previous confinement without Eclampsia.

1907. May 15th. Was confined about 5 p.m. Single child alive. Labour very precipitate - only two or three pains having occurred. No prodromata as, headache, blindness observed. I was informed, however, that she had suffered from severe sickness during the whole term of pregnancy up to time of labour. I arrived on the scene about 5.30 p.m., found everything in order and patient apparently remarkably well. I left shortly after six.

About 6.30 she was seized with a convulsion from which she recovered and shortly afterwards regained consciousness.

Between this and 10 p.m. three such convulsions followed each succeeded by short periods of coma. Having a considerable amount of work to go through elsewhere, I was unaware of the occurrence of the Eclampsia till my return to patient's house about 10 p.m. She was then recovering from her third fit. Having no suspicion beforehand of what was awaiting me and being a long distance from my own house and having no hypodermic syringe, also considering the circumstance that she soon regained consciousness and was able to swallow, after remaining for about one hour and there being no sign of further convulsions, I for

the time being prescribed Potass Bromide in 25 grains every four or five hours and enjoined a strictly milk diet. About 4 a.m. next morning, however, she was seized with another convulsion and another about 9 a.m. This was the last convulsion. There was a slight degree of oedema. Urine was found to be loaded with albumen.

The albuminuria rapidly disappeared and in about 10 days no trace was left. About the third day of the puerperium a purpuric rash appeared in the face and rapidly spread over the whole body. This continued for about one week, after which it gradually faded away. She made a good recovery and rose about the 14th day.

She again became pregnant about the latter end of 1908. About one month before her confinement, which took place in August, on testing her urine I found it contained a considerable quantity of albumen. By adopting the usual prophylactic measures along with a strictly milk diet, the albuminuria was soon reduced to a minimum. On the 9th August labour occurred precipitately as on the last occasion. There was no Eclampsia. The urine in a few days was entirely free from albumen and after a puerperium of about 9 or 10 days, she was able to get out of bed and made a good recovery.

I am indebted to Dr. Cumming for the next three cases.

Mrs B. Age 30, primipara. 1910. About middle of January. Then about seven months pregnant. Urine examined found to be free from albumin. She was instructed to call again with another sample of urine but disobeyed.

About Feb. 3rd, noticed oedema of legs, but not any striking decrease of quantity of urine passed.

Mar. 3rd. Labour commenced about 11 a.m. Dr. Cumming summoned about 7.30 p.m. Labour finished normally about 9.30 p.m. Dr. Cumming left about one hour later, patient being then apparently well except for oedema of legs. She continued in the same outwardly satisfactory condition all night.

Mar. 4th, 11.30. Seized with a convulsion. Dr. Cumming called a few minutes later, but not having a hypodermic with him went to fetch one.

From 11.30 a.m. to 12.30 p.m. she had 5 fits. About 12.30 p.m. ½ grain Morphia injected hypodermically. After this no more fits occurred. 2 hours later ¼ grain Morphia administered and two hours afterwards again another ¼ grain Morphia. She then slept soundly all night. Urine drawn off by catheter was found to contain much albumin. The precipitate obtained by boiling and nitric acid stood about half the height of the whole quantity of urine in the test tube.

Previous to confinement she was on no special diet, but after the convulsions she was kept on a strictly milk diet for 10 days, during which time the urine contained albumin at first in considerable quantity but gradually through that period decreasing till about the tenth day it had almost entirely disappeared. After this time she was put on farinaceous food and later on on white flesh. At the present date there is still a slight trace of albumin.

The foetus was born alive apparently at full term, well nourished and quite active, apparently about 8 lbs. in weight, and since then has done well. No evidence that foetus imbibed any toxin. ~~Dr. Cumming considers this a sign that the malady had not been long existent in the maternal system.~~

Dr. Cumming's Second Case.

Mrs D. Age 28 years. Primipara. Confined February 28th, 1910.

Two months previously oedema of face observed. Urine found to contain much albumin. She was put on a modified diet and ordered an alkaline mixture containing potass acetate 25 grains thrice daily. No reduction of albumin followed this treatment. She was then put on a diet of milk alone and diuretin in doses of 10 grains administered night and morning. The result now was that urine showed only a slight trace of albumin. This satisfactory condition remained until she was allowed out of bed and attempted stronger dieting which brought back the excess of albuminuria. She was then put back to bed and the strictly milk diet with diuretin was resumed. This was again followed with the satisfactory result of diminution of the albuminuria till only a trace was present, and continued in this condition until labour took place on February 29th 1910. Labour normal; foetus thin, ill-nourished, weighing 4½ lbs. which would seem to indicate that the tendency to convulsions is decreased in proportion to the lowering of the foetal metabolism.

Dr. Cumming's Third Case.

Mrs M. Age about 38 years. Primipara.

Confinement due about May 9th, 1908.

April 13th, 11 p.m. Dr. Cumming, on being summoned to attend, finds her in a convulsion, and was informed that she had been similarly seized shortly before.

Examination revealed os fully dilated and head presentation. Under chloroform anaesthesia he applied forceps and delivered a living child with comparative ease. He then discovered that a second foetus was present in utero. On applying pressure suprapubically, he was able to expel second child along with double placenta and membranes all in one, about twenty minutes after first. The second child was dead. Urine drawn off by catheter found to be loaded with albumin. She was kept on a diet of milk alone for a few days, during which period albuminuria gradually cleared off till ultimately no trace was left. She made a good recovery.

The outstanding feature here is the association of Eclampsia (1) with twins, (2) with the advanced age, (3) primipara.

Summary.

~~I. Reasons for adopting title in view of Dr. Muhlendie's remark.~~

I ~~II~~. Introduction - referring to the circumstance that certain risks and dangers incidental to pregnancy are now much more effectually dealt with, but that Eclampsia still confronts us as a serious factor to be encountered.

II ~~III~~. Frequency of its occurrence taken from statistics of various sources.

III ~~IV~~. Nature of the Malady:-

(a) Older views.

(b) Modern views.

Older Views: (1) Morbid cerebral condition; (2) Exalted nervous tension; (3) Bacterial theory; (4) Renal inadequacy alone; (5) Retention of urinary constituents (urinaemia); (6) Acute nephritis.

Modern Views: (1) Thyroid inadequacy; (2) Transmission of foetal products into maternal circulation; (3) Placental theory,- (a) Syncytium, (b) placental cell autolysis, (c) placental nucleo-proteid.

IV. Reasons against placental cell theory:-

(a) Ascertained facts derived from investigation not sufficient to warrant its assumption.

(b) The circumstance that certain cases of Eclampsia occur after expulsion of placenta.

V ~~VI~~. Probable immediate or exciting causes of Eclampsia:-

(a) Toxin or combination of toxins traceable to ~~ammo~~ Amino-acids circulating in blood through hepatic failure to transform them into urea.

(b) Mechanical pressure on brain of either emboli, haemorrhages or aneurisms.

(c) Important share in above processes by both thyroid and suprarenal functions.

VI

~~VII~~. Attempts, from a clinical survey of the most outstanding physiological and pathological features occurring during pregnancy, to prove that the existence in the blood of those ~~Ammo~~-Acids, emboli, etc., are not the outcome of placental derivatives, but are due solely to excessive metabolic changes taking place in the maternal system, Autolysis especially of the liver playing a very important part.

VII

~~VIII~~. Symptoms.

VIII ~~IX~~. Treatment, a short summary of which follows the more detailed description.

IX. Cases.

---

x Amino